JAFIB
Case Report
Journal of Atrial Fibrillation

www. jafib.com

Atrial Arrhythmias in Chagas' Disease

Jorge O. Diaz[1], Camilo A. Rueda[2]

[1] *Central Florida Regional Hospital, Sanford, Fl; Fundación Cardiovascular de Colombia, Bucaramanga, Colombia.*

[2] *Pontificia Universidad Javeriana, Bogota, Colombia.*

Abstract

There is extensive knowledge in the ablation of ventricular arrhythmias in patients with Chagas' cardiomyopathy, as well as specific characteristics of the arrhythmogenic substrate, and the need for an epicardial approach to achieve success. On the contrary, no publications currently address the ablation of atrial fibrillation in these patients or the characteristics of the arrhythmia such as the successful ablation sites. Our two case reports highlight the association of Chagas' disease with atrial arrhythmias, the tailored approach that was needed to give an appropriate treatment and a discussion of our current knowledge of Chagas' cardiomyopathy as a substrate for atrial arrhythmias.

Introduction

Since the first description of pulmonary vein depolarizations as triggers of atrial fibrillation (AF) by Haïssaguerre, there has been a widespread use of ablation techniques for the treatment of paroxysmal atrial fibrillation. Furthermore, the techniques have been modified and have also included patients with persistent atrial fibrillation as candidates for ablation procedures[1]. In most of the industrialized countries, atrial fibrillation is the result of many etiologies, including hypertensive heart disease, obesity, obstructive sleep apnea, heart failure and coronary artery disease. This is also valid for third world countries; however, other causes of heart disease, such as malnutrition and parasitic infections including Chagas' disease are of importance, especially in areas where there is a high prevalence[2].

Pulmonary vein isolation (PVI) is the cornerstone for all the existing ablation techniques targeting paroxysmal atrial fibrillation. PVI has been extrapolated to persistent atrial fibrillation, with limited results, due to the presence of atrial fibrosis and other abnormalities acting as substrate. Therefore, modification of the arrhythmogenic substrate has been postulated, with better, although not satisfactory results.

Chagas' cardiomyopathy manifests as conduction system disease, ventricular arrhythmias and sudden cardiac death. There is extensive knowledge in the ablation of ventricular arrhythmias in these population, as well as specific characteristics of the arrhythmogenic substrate, and the need for an epicardial approach to achieve success. On the contrary, no publications currently address the ablation of atrial fibrillation in these patients or the characteristics of the arrhythmia such as successful ablation sites.

We present two cases of atrial arrhythmias in patients with Chagas' cardiomyopathy in which a conventional approach would have been unsuccessful due to the specific characteristics of the arrhythmia, in regards to the origin, substrate and successful ablation sites.

Key Words

Atrial, Arrythmias, Chagas, Atrial Fibrillation

Corresponding Author
Jorge O. Diaz,
1331 South International Parkway Suite 1261, Lake Mary, FL, 32746

Case 1

A 56 years old male patient with a history of Chagas' cardiomyopathy, ventricular tachycardia and implantation of a dual chamber defibrillator, experienced inappropriate shocks for AF, despite treatment with beta-blockers and amiodarone. Echocardiogram showed an ejection fraction of 30%, with left and right atrial areas measured at end systole of 17 and 19 cm^2, respectively. A duo decapolar catheter was placed along the Crista Terminalis (CT) and in the Coronary Sinus (CS). A quadripolar catheter was placed in the His bundle position. Single transeptal technique was used. St. Jude Agilis steerable sheath, a Reflection Spiral decapolar catheter and a Cool Flex ablation catheter, were used for 3D mapping and ablation purposes. The initial approach included Wide Area Circumferential Ablation (WACA) followed by elimination of Complex Fractionated Atrial Electrograms (CFAE) and linear ablation of the roof, posterior wall of the left atrium and the mitral isthmus. Despite demonstration of pulmonary vein and posterior wall isolation as well as complete electrical silence in the left atrium [Figure 1], AF persisted. Fibrillatory activity persisted in the channels recording the Right Atrium (RA)[Figure 2].

Therefore, we proceeded with CFAE mapping of this chamber. CFAE were eliminated in the RA, resulting in organization of tachycardia into an atrial flutter with a cycle length of 290 msec [Figure 3], successfully entrained in the lower lateral wall of the RA [Figure 4]. Interestingly, there was a large area of scar encompassing the posterior and lateral wall of the RA. An ablation line from the

superior vena cava to the inferior vena cava further increased the cycle length and changed the activation, findings consistent with an atrial tachycardia from the low CT [Figure 5], which was successfully terminated with restoration of sinus rhythm. Clinically, the patient

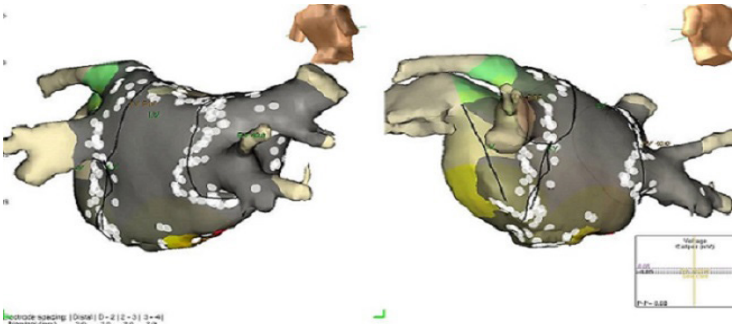

Figure 1: PVIand Posterior Wall isolation.

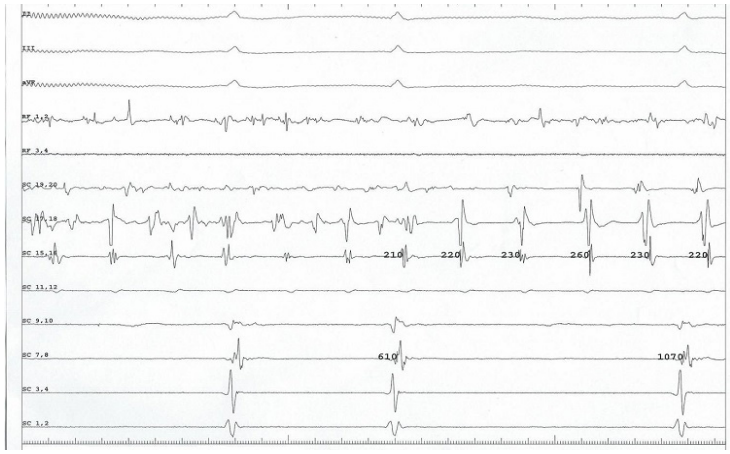

Figure 2: AF continues in the right atrium.

has been asymptomatic and interrogation of his device has not shown any recurrent atrial arrhythmias.

Case 2

A 63 years old male patient who presented with sustained palpitations due to a narrow complex tachycardia with hemodynamic instability. Echocardiogram revealed an ejection fraction of 55%, mild

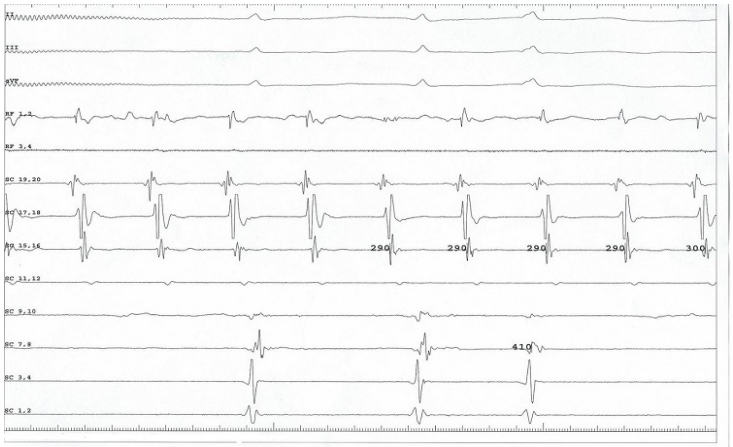

Figure 3: Organization of fibrillatory activity into atrial flutter.

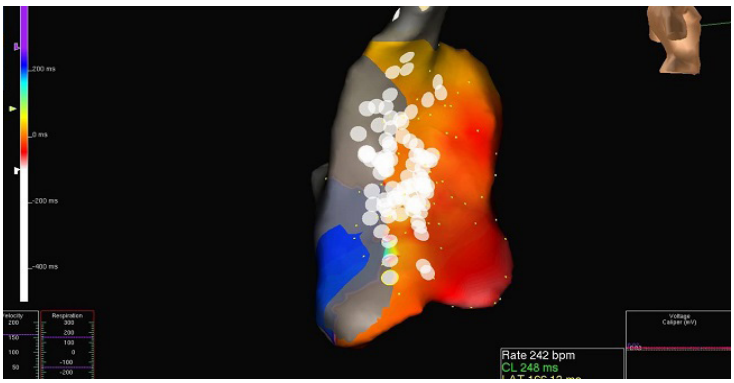

Figure 4: Right Atrium Activation Map and Ablation.

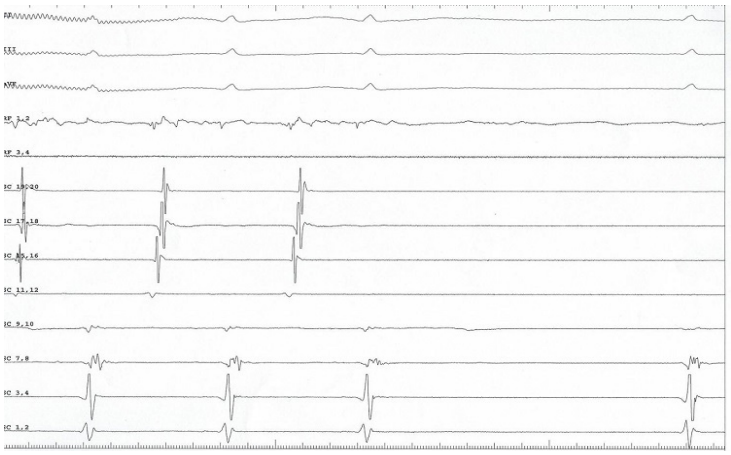

Figure 5: Termination of Right Atrial Tachycardia

left ventricular dilatation, moderate aortic insufficiency and mild biatrial enlargement. The left atrium area at end systole is 27 cm2. Antibodies against T. cruziwere positive by enzyme immunosorbent assay. Initial ECG is consistent with atrial flutter with a cycle length of approximately 280msec [Figure 6]. A duo decapolar catheter was placed along the CT and in CS. A quadripolar catheter was placed in the His bundle position but later was placed in the distal CS. A St. Jude Cool Flex catheter was used for mapping and ablation purposes. During anesthesia induction, the patient became hemodynamically unstable, therefore he was cardioverted. After his vital signs

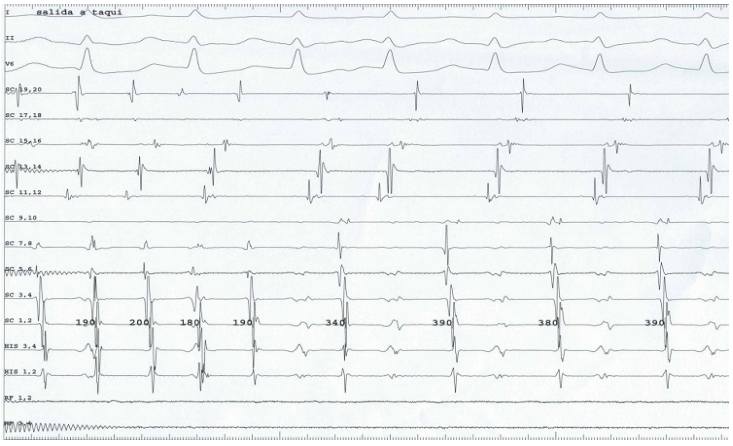

Figure 6: EGMs of Atrial Flutter

normalized, he was easily induced into a fast-atrial flutter with a cycle length of 180 to 200msec. Occasionally, there were episodes that resemble AF. An initial cavotricuspid isthmus ablation line did not impact the tachycardia;therefore, we proceeded with activation mapping. An ablation line from the superior vena cava to the inferior vena cava further increased the cycle length and subsequently terminated the tachycardia [Figure 7]. Isoproterenol was given up to 20 mcg/min and aggressive pacing maneuvers were performed with no induction on any other atrial arrhythmias.

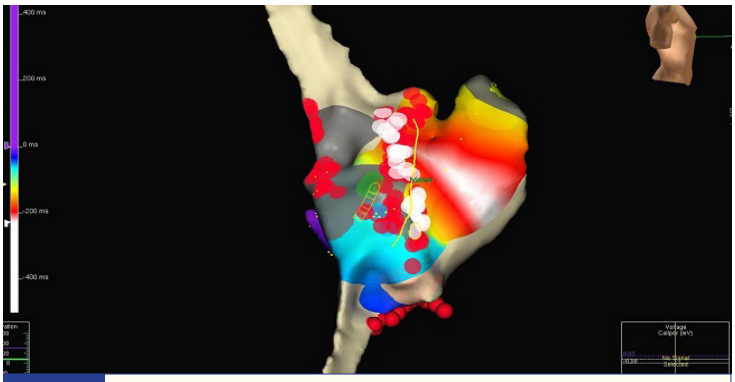

Figure 7: Right Atrium Activation Map and Ablation Line

Discussion

CD affects 8-12 million people in Latin America and for the US it is estimated that 300,000 people suffer of chronic Chagas' disease [3,4]. Although the pathogenesis of CD is not completely understood, it is known that reparative fibrosis is the hallmark of chronic CD[3]. Reparative fibrosis promotes the development of dilated cardiomyopathy and usually affectsthe ventricular myocardium. Furthermore, the fibrotic process generally becomes the substrate for ventricular, but its association with the development of AF is controversial[4].

Over the past 20 years, there has been an explosion of knowledge of the pathophysiology of AF. Clinical AF results from an interaction between triggers and sustaining mechanisms, acting as substrates, composed of electrical and/or structural components. AF is often triggered by ectopy from the pulmonary veins. Pulmonary vein triggers may be promoted by activity from nearby ganglionatedplexi, and relate to structural-functional abnormalities at the junction between left atrial and pulmonary vein tissue[5,8]. Although pulmonary veins accounts for a majority of cases of AF, less defined atrial sites can have an association with atrial fibrillation. This may explain, in part, why some patients with presumed paroxysmal AF respond poorly where as others with presumed persistent AF respond favorably to pulmonary vein isolation alone. Furthermore, recent data suggests that atrial fibrosis, a marker of the underlying substrate, can be visualized using cardiac magnetic resonance imaging and be used to individualize the strategy for catheter ablation[9].

The reported cases illustrate how CD can produce unusual arrhythmogenic foci and substrate for the development of AF. The first case shows how traditional PVI, although successfully accomplished, failed and AF persisted in the RA. Furthermore, the second case illustrates how atrial fibrosis developed in the posterior wall of the RA and acted as substrate for a reentrant atrial tachycardia. This explains why the arrhythmia persisted after the traditional approach of acavotricuspid isthmus ablation. In general, these findings support the hypothesis that fibrosis and Chagasic cardiomyopathy serve as substrate for multiple atrial arrhythmias and that traditional ablation approaches can fail to treat atrial arrhythmias in these individuals.

Finally, we believe that AF should be classified in a more utilitarian and precise fashion, perhaps using terms that assign both etiologic and mechanic information when appropriate.

Conclusion

CD affects the heart tissue and promotes the development of fibrosis and cardiomyopathy, which is the substrate for arrhythmogenic foci. Clinicians should be aware of these abnormalities and be prepared to tailor the ablation procedure accordingly.

References

1. Wazni O, Wilkoff B, Saliba W. Catheter ablation for atrial fibrillation. N. Engl. J. Med. 2011;365 (24):2296–304.
2. Pereira MC, Dones W, Morillo CA, EncinaJuan Justiniano, RibeiroAntônio Luiz. Chagas disease: an overview of clinical and epidemiological aspects. J. Am. Coll. Cardiol. 2013;62 (9):767–76.
3. E-Journal of Cardiology Practice Mora G. Chagas cardiomyopathy.2016 August 20, 2018; Vol. 14, N° 31. Available from: https://www.escardio.org/Journals/E-Journal-of-Cardiology-Practice/Volume-14/Chagas-cardiomyopathy.
4. Cardoso R, Garcia D, Fernandes G, He LI, Lichtenberger P, Viles-Gonzalez J, Coffey JO, Mitrani RD. The Prevalence of Atrial Fibrillation and Conduction Abnormalities in Chagas' Disease: A Meta-Analysis. J. Cardiovasc. Electrophysiol. 2016;27 (2):161–9.
5. Calkins H, Kuck KH, Cappato R, Brugada J, Camm AJ, Chen SA, Crijns HJ, Damiano RJ, Davies DW, Di MJ, Edgerton J, Ellenbogen K, Ezekowitz MD, Haines DE, Haissaguerre M, Hindricks G, Iesaka Y, Jackman W, Jalife J, JaisPierre, Kalman J, Keane D, Kim YH, Kirchhof P, Klein G, Kottkamp H, Kumagai K, Lindsay BD, Mansour M, Marchlinski FE, Mc CP M, Mont JL, Morady F, Nademanee K, Nakagawa H, Natale A, Nattel S, Packer DL, Pappone C, Prystowsky E, Raviele A, Reddy V, Ruskin JN, Shemin RJ, Tsao HM, Wilber D. 2012 HRS/EHRA/ECAS expert consensus statement on catheter and surgical ablation of atrial fibrillation: recommendations for patient selection, procedural techniques, patient management and follow-up, definitions, endpoints, and research trial design: a report of the Heart Rhythm Society (HRS) Task Force on Catheter and Surgical Ablation of Atrial Fibrillation. Developed in partnership with the European Heart Rhythm Association (EHRA), a registered branch of the European Society of Cardiology (ESC) and the European Cardiac Arrhythmia Society (ECAS); and in collaboration with the American College of Cardiology (ACC), American Heart Association (AHA), the Asia Pacific Heart Rhythm Society (APHRS), and the Society of Thoracic Surgeons (STS). Endorsed by the governing bodies of the American College of Cardiology Foundation, the American Heart Association, the European Cardiac Arrhythmia Society, the European Heart Rhythm Association, the Society of Thoracic Surgeons, the Asia Pacific Heart Rhythm Society, and the Heart Rhythm Society. Heart Rhythm. 2012;9 (4):632–696.e21.
6. Narayan SM, Baykaner T, Clopton P, Schricker A, Lalani GG, Krummen DE, Shivkumar K, Miller JM. Ablation of rotor and focal sources reduces late recurrence of atrial fibrillation compared with trigger ablation alone: extended follow-up of the CONFIRM trial (Conventional Ablation for Atrial Fibrillation With or Without Focal Impulse and Rotor Modulation). J. Am. Coll. Cardiol. 2014;63 (17):1761–8.

7. Marrouche NF, Wilber D, Hindricks G, Jais P, Akoum N, Marchlinski F, Kholmovski E, Burgon N, Hu N, Mont L, Deneke T, Duytschaever M, Neumann T, Mansour M, Mahnkopf C, Herweg B, Daoud E, Wissner E, Bansmann P, Brachmann J. Association of atrial tissue fibrosis identified by delayed enhancement MRI and atrial fibrillation catheter ablation: the DECAAF study. JAMA. 2014;311 (5):498–506.
8. Akoum N, Daccarett M, Mc GC, Segerson N, Vergara G, Kuppahally S, Badger T, Burgon N, Haslam T, Kholmovski E, Macleod R, Marrouche N. Atrial fibrosis helps select the appropriate patient and strategy in catheter ablation of atrial fibrillation: a DE-MRI guided approach. J. Cardiovasc. Electrophysiol. 2011;22 (1):16–22.
9. Segerson NM, Daccarett M, Badger TJ, Shabaan A, Akoum N, Fish EN, Rao S, Burgon NS, Adjei PY, Kholmovski E, Vijayakumar S, Di Bella EV, Mac LR, Marrouche NF. Magnetic resonance imaging-confirmed ablative debulking of the left atrial posterior wall and septum for treatment of persistent atrial fibrillation: rationale and initial experience. J. Cardiovasc. Electrophysiol. 2010;21 (2):126–32.